

**Amendments to the Claims:**

This listing of claims will replace all prior versions, and listings of claims in the application.

**Listing of Claims:**

1. (Canceled)

2. (Previously Presented) A method according to claim 17 wherein the antagonist of the CB1 receptor is a specific antagonist of the CB1 receptor.

3-11. (Canceled)

12. (Previously Presented) A method according to claim 17 wherein the CB1 receptor is selected from the group consisting of:

a) a protein having an amino acid sequence comprising SEQ ID NO: 1 or a portion of SEQ ID NO:1, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

b) a protein having an amino acid sequence comprising SEQ ID NO: 2 or a portion of SEQ ID NO: 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

c) an allele of the protein having the amino acid sequence of SEQ ID NO:1 or SEQ ID NO: 2, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal;

d) a protein having the amino acid sequence of SEQ ID NO:1 with a Phenylalanine to Leucine substitution at position 200; and/or an Isoleucine to Valine substitution at position 216; and/or a Valine to Alanine substitution at position 246;

e) a protein having the amino acid sequence of SEQ ID NO: 2 with a Phenylalanine to Leucine substitution at position 139; and/or an Isoleucine to Valine substitution at position 155; and/or a Valine to Alanine substitution at position 185; and

f) a protein comprising the amino acid sequences of SEQ ID NO: 3, SEQ ID NO: 4, SEQ ID NO:5, SEQ ID NO: 6, SEQ ID NO: 7, SEQ ID NO: 8 and SEQ ID NO: 9 or amino acid

sequences 80% homologous to these, said protein having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

13. (Previously Presented) A method according to claim 17 wherein the CB1 receptor is a protein having a homology at the amino acid level with SEQ ID NO: 1 of at least 45%, having the biological function of a G protein-coupled cellular receptor, capable of binding THC and transducing a cellular signal.

14. (Previously Presented) A method according to the preceding claim 13 wherein the homology is at least 60%.

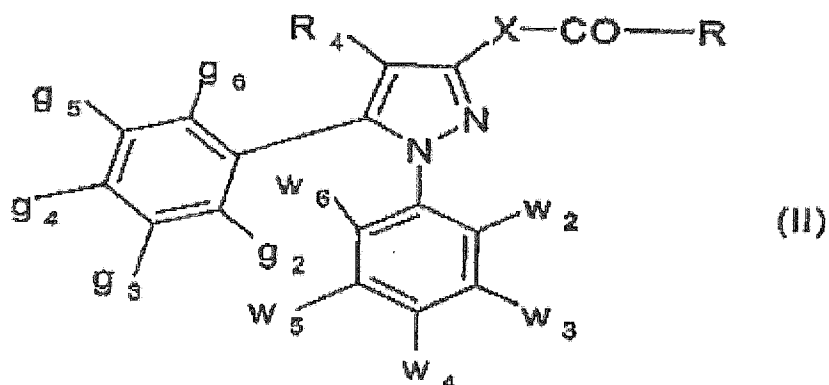
15. (Previously Presented) A method according to claim 17 wherein the daily dosage of CB1 receptor antagonist is from 0.01mg to 500mg.

16. (Canceled)

17. (Currently Amended) A method of treatment of hepatic diseases in a mammal ~~comprising~~consisting essentially of:

administering a therapeutically effective amount of at least one CB1 receptor antagonist to a mammal in need thereof, wherein the CB1 receptor antagonist is a compound of the formula II or one of its pharmaceutically acceptable salt, in which  $g_2$ ,  $g_3$ ,  $g_4$ ,  $g_5$  and  $g_6$  and  $w_2$ ,  $w_3$ ,  $w_4$ ,  $w_5$  and  $w_6$  are identical or different and are independently hydrogen, a chlorine or bromine atom, a (C<sub>1</sub>-C<sub>3</sub>) alkyl, a (C<sub>1</sub>-C<sub>3</sub>) alkoxy, a trifluoromethyl or a nitro group and  $g_4$  is optionally a phenyl group;  $R_4$  is hydrogen or a (C<sub>1</sub>-C<sub>3</sub>) alkyl; X is either a direct bond or a group  $-(CH_2)_x-N(R_3)-$ , in which  $R_3$  is hydrogen or a (C<sub>1</sub>-C<sub>3</sub>) alkyl and x is zero or one; R is: a group  $-NR_1R_2$  in which  $R_1$  and  $R_2$  are independently a (C<sub>1</sub>-C<sub>6</sub>)-alkyl; an non-aromatic (C<sub>3</sub>-C<sub>15</sub>) carbocyclic radical which is optionally substituted, said substituent (s) being other than a substituted carbonyl; an amino(C<sub>1</sub>-C<sub>4</sub>) alkyl group in which the amino is optionally disubstituted by a (C<sub>1</sub>-C<sub>3</sub>) alkyl; a cycloalkyl (C<sub>1</sub>-C<sub>3</sub>) alkyl in which the cycloalkyl is C<sub>3</sub>-C<sub>12</sub>; a phenyl which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C<sub>1</sub>-C<sub>5</sub>) alkyl or by a (C<sub>1</sub>-C) alkoxy; a phenyl (C<sub>1</sub>-C<sub>3</sub>) alkyl ; a diphenyl (C<sub>1</sub>-C<sub>3</sub>) alkyl; a naphthyl; an anthracenyl; a saturated 5-to 8-membered heterocyclic radical which is unsubstituted or substituted by a (C<sub>1</sub>-C<sub>3</sub>) alkyl, by a hydroxyl or by a benzyl; a 1-adamantylmethyl; an aromatic heterocycle which is

unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C<sub>1</sub>-C<sub>5</sub>) alkyl or by a (C<sub>1</sub>-C<sub>5</sub>) alkoxy; a (C<sub>1</sub>-C<sub>5</sub>) alkyl which is substituted by an aromatic heterocycle which is unsubstituted or monosubstituted or polysubstituted by a halogen, by a (C<sub>1</sub>-C<sub>5</sub>) alkyl or by a (C<sub>1</sub>-C<sub>5</sub>) alkoxy; or else R<sub>1</sub> is hydrogen and R<sub>2</sub> is as defined above; or else R<sub>1</sub> and R<sub>2</sub> form a saturated 5-to 8-membered heterocyclic radical with the nitrogen atom to which they are bonded, said heterocyclic radical being other than morpholine when w<sub>2</sub>, w<sub>3</sub>, w<sub>4</sub>, w<sub>5</sub>, w<sub>6</sub>, g<sub>2</sub>, g<sub>3</sub>, g<sub>4</sub>, g<sub>5</sub> and g<sub>6</sub> are all hydrogen; a group R<sub>2</sub> as defined above when X is -(CH<sub>2</sub>)<sub>x</sub>N(R<sub>3</sub>)-; a group R<sub>5</sub> when X is a direct bond, R<sub>5</sub> being a (C<sub>1</sub>-C<sub>3</sub>) alkyl; a (C<sub>3</sub>-C<sub>12</sub>) cycloalkyl which is unsubstituted or substituted by a (C<sub>1</sub>-C<sub>5</sub>) alkyl; a phenyl(C<sub>1</sub>-C<sub>3</sub>) alkyl which is unsubstituted or substituted by a halogen or by a (C<sub>1</sub>-C<sub>5</sub>) alkyl; a cycloalkyl (C<sub>1</sub>-C<sub>3</sub>) alkyl in which the cycloalkyl is C<sub>1</sub>-C<sub>12</sub> and is unsubstituted or substituted by a (C<sub>1</sub>-C<sub>5</sub>) alkyl; or a 2-norbornylmethyl.



18. (Cancelled)

19. (Previously Presented) A method according to claim 17 wherein the CB1 receptor antagonist is N-piperidono-3-pyrazolecarboxamide or one of its pharmaceutically acceptable salt.

20. (Withdrawn) A method according to claim 17 wherein the CB1 receptor antagonist is N-piperidino-5-(4-bromophenyl)-1-(2,4-dichlorophenyl)-4-ethylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

21. (Previously Presented) A method according to claim 17 wherein the CB1 receptor antagonist is N-piperidino-5- (4-chlorophenyl)-1- (2, 4-dichlorophenyl) -4-methylpyrazole-3-carboxamide or one of its pharmaceutically acceptable salt.

22. (Previously Presented) A method according to claim 17 wherein the hepatic disease is liver fibrosis.

23. (Previously Presented) A method according to claim 17 wherein the hepatic disease is alcoholic liver cirrhosis.

24. (Previously Presented) A method according to claim 17 wherein the hepatic disease is chronic viral hepatitis.

25. (Previously Presented) A method according to claim 17 wherein the hepatic disease is non-alcoholic steatohepatitis.

26. (Previously Presented) A method according to claim 17 wherein the hepatic disease is primary liver cancer.

27. (Previously Presented) A method according to claim 17 wherein the daily dosage of CB1 receptor antagonist is from 1mg to 100mg.